Inflammation

By

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INFLAMMATION

Definition:

Response of living tissue to injurious agent.

The response consists of a series of vascular, lymphatic and local tissue changes.

Aim of inflammation:

1. Destroy, dilute, remove or localize injurious agents.
2. replacement of the damaged tissue by a new healthy one.

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-itis

Appendicitis

Meningitis

Tonsillitis

Nephritis

Myocarditis

Causes of inflammation

The injurious agents which cause inflammation are called irritants, these may be:

(A) Living irritants

Bacteria, viruses, fungi and parasites.

(B) Non living irritants: Include

- **i- Physical irritants**
- **ii- Chemical irritants**
- **iii- Necrotic tissue**
- **iv- Immune mechanism** (Ag/Ab reaction)

Types of inflammation

According to the onset, severity and duration of irritation, it is classified into:

(1) Acute inflammation:

- It is of sudden onset, short duration.
- Caused by strong irritant e.g. abscess caused by staphylococci
- Followed repair

(2) Chronic inflammation:

- It is of gradual onset, longer duration.
- Caused by mild persistent irritant e.g. Tuberculosis
- Associated with repair

Acute Inflammation

Morphological changes in acute inflammation

I) Vascular changes.

II) Exudative changes.

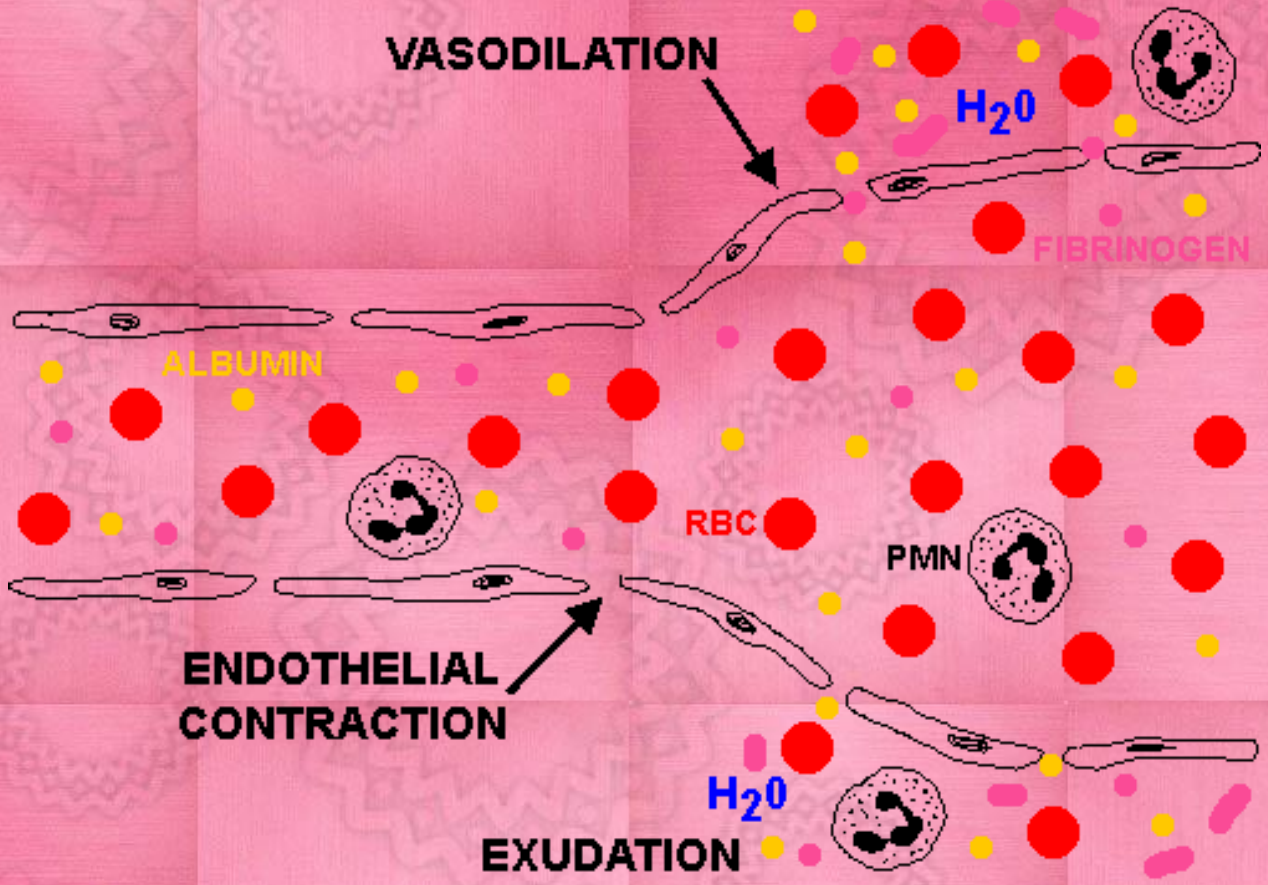
III) Local tissue changes.

I. Vascular changes

A) Transient vasoconstriction of the arterioles lasting for few seconds followed by Permanent vasodilatation.

B) Increase vascular permeability.

C) Temporary acceleration of blood flow followed later by gradual slowing.



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II- Exudative phenomenon

(A) Fluid component (fluid exudate)

Accumulation of extra cellular fluid at the area of inflammation to dilute the irritant specially chemical and bacterial toxins. It brings antibodies to the site of inflammation

(B) Cellular component (cellular exudate)

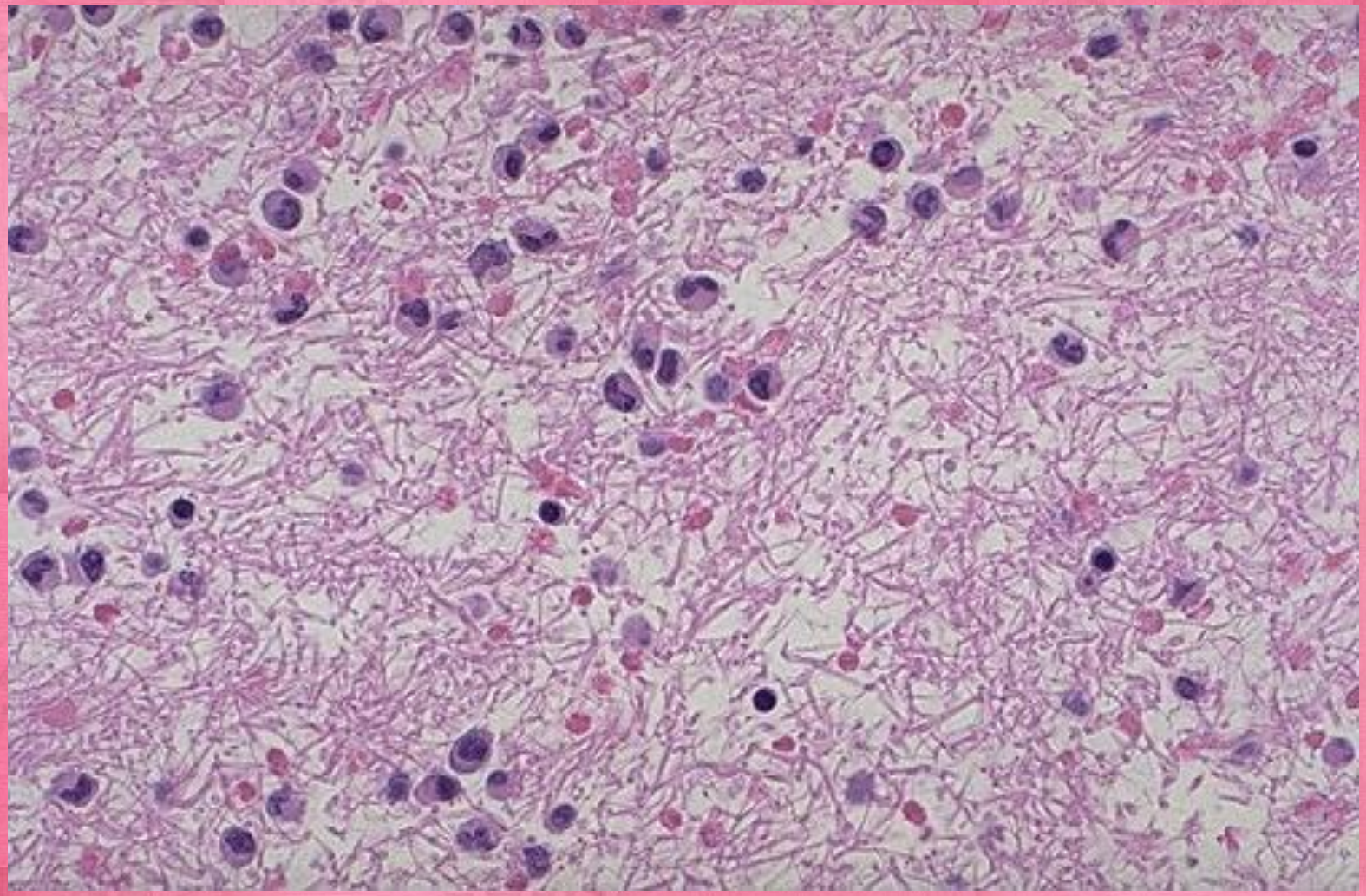
Escape of leucocytes (neutrophils, macrophages) outside the circulation for destruction of microorganisms and necrotic debris.

Cellular exudate:

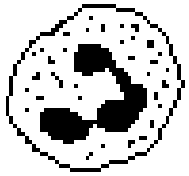
Steps;

1. Margination: leucocytes move from the center to the margin (periphery) of the blood stream
2. Emigration: leucocytes escape out the blood vessels through the inter-endothelial gaps.
3. Chemotaxis: It is the directed movement of leucocytes towards the irritant
4. Phagocytosis: engulfment and destruction of the irritant.

Exudate



MARGINATION



III-Local Tissue Changes

- The irritant is at a maximum concentration in the center of the inflammatory area where it produced necrosis (death of group of cells).
- In the surrounding area, it is of lesser intensity producing degeneration (sick cell).
- Necrotic cells and degenerated cells release chemical substances called chemical mediators which help the vascular changes.

Systemic effects of acute inflammation

1. **Fever:** due to cytokines especially (interleukin 1 (IL-1) and tumor necrosis factor (TNF).
2. **Leucocytosis:** due to IL-I and TNF that stimulate bone marrow..

Chemical mediators of acute inflammation

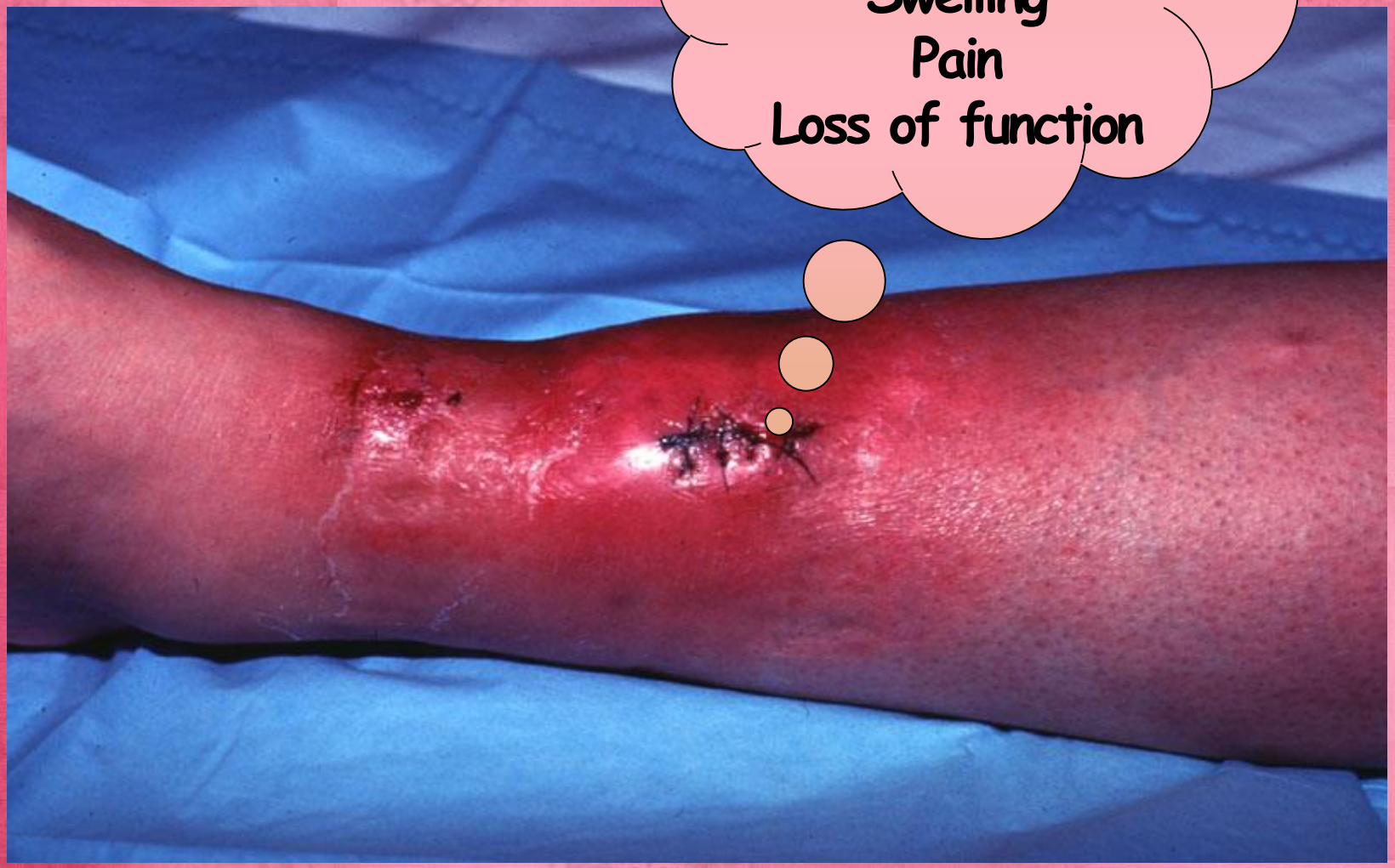
These are mediator substance that influence the inflammatory process.

- Exogenous mediators of microbial origin (as peptides of E.coli are chemotactic).
- Endogenous mediators from cells or plasma.

The main actions of chemical mediators are:

- Vascular dilatation: Caused e.g. by histamine.
- Increased vascular permeability: As histamine and kinins.
- Chemotaxis: Caused by Leukotrienes, lysosomal components and C5a.
- Pain: as bradykinin.

Cardinal Signs



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Systemic effects of acute inflammation

(1) Fever: caused by pyrogens (i.e. fever producing substances):

- Exogenous: released from bacteria and fungi.
- Endogenous: cytokines as interleukin 1 (IL-1) and tumor necrosis factor (TNF).

(2) Leucocytosis: caused by IL-1 and TNF that stimulate release of leucocytes from bone marrow.

- **Types of acute inflammation**

I- Suppurative inflammation.

II- Non-Suppurative inflammation.

Suppurative inflammation

A- Localized

1- Abscess: A localized suppurative inflammation resulting in the formation of an irregular cavity containing pus

2- Furuncle (Boil)

3- Carbuncle

B- Diffuse: e.g. Cellulitis, suppurative appendicitis

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Non-Suppurative inflammation

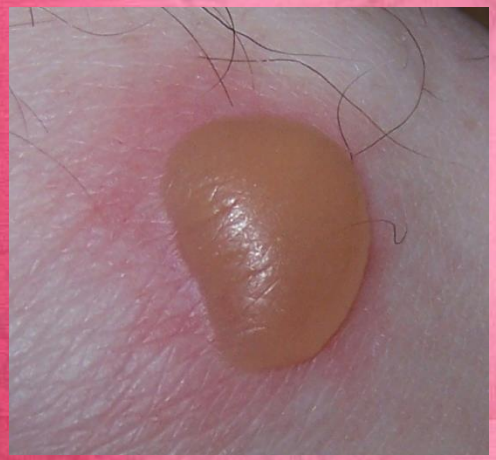
Excess erythrocytes (Hemorrhagic)

Excess fluid exudate

- Serous
- Serofibrinous & Fibrinous
- Catarrhal

Excess necrosis

- Necrotizing
- Membranous
- Allergic



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Non-Suppurative inflammation

1. **Catarrhal inflammation:** common cold (= coryza).
2. **Membranous (pseudomembranous) inflammation:** Diphtheria & bacillary dysentery).
3. **Serofibrinous inflammation** serous sacs (pleura, pericardium and peritoneum).
4. **Fibrinous inflammation:** in lobar pneumonia.
5. **Serous inflammation** as herpes simplex and burn where watery vesicles are seen.
6. **Hemorrhagic inflammation** as Small pox, meningococci.
7. **Necrotizing inflammation**; extensive tissue necrosis e.g. Infective gangrene in the lungs etc.
8. **Allergic inflammation** in hypersensitivity reactions..

Chronic Inflammation

(1) Chronic non specific inflammation:
Irritants cannot be identified, follow acute inflammation as chronic abscess.
(2) Chronic specific inflammation (Granuloma):
Each irritant produces a specific reaction; the etiological factor can be identified.

Types of Granuloma

1. Bacterial: e.g. tuberculosis, leprosy and syphilis.
2. Viral: lymphogranuloma inguinale
3. Parasitic: e.g. Bilharziasis.
4. Fungal: Actinomycosis.
5. Allergic granuloma: as Rheumatic fever.
6. Foreign body granuloma: around foreign bodies as asbestos, silica..
7. Granulomas of unknown cause: as sarcoidosis

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	Acute inflammation	Chronic inflammation
Irritant	Mild, moderate or severe	Mild
Onet	Sudden	Gradual
Duration	Short (days, weeks)	Long (months, years)
Vascular changes	Marked	Mild
bl. Vessels	Thin, dilated congested capillaries	Thick walled arterioles (endarteritis)
Inflammatory cells	Neutrophils, pus and macrophages	Lymphocytes, plasma cells, macrophages and giant cells.
Fibrosis	Absent or mild	Marked

Thank you

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